

Vertical Muscles Transposition with Medial Rectus Botulinum Toxin Injection for Abducens Nerve Palsy

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Abstract. Finding an alternative to ipsilateral medial rectus recession when performing superior and inferior rectus transposition surgery; thus eliminating the risk of anterior segment ischemia and overcorrection. Fourteen patients with sixth nerve palsy of more than 8 month duration were included in the study. Ages ranged between 6 to 76 years old. All the patients underwent full tendon transposition of the Superior and Inferior rectus muscles to the lateral rectus muscle insertion and injection of the ipsilateral medial rectus muscle with botulinum toxin. Two patients had a significant esotropia (more than 8 prisms) and were advised to have a recession surgery for the ipsilateral medial rectus. Six patients had a non significant esotropia, 4 patients were ortho and 2 were exotropic (less than 12 prisms). Transposition procedures are the best option for treating abducens nerve palsy and almost always require an ipsilateral medial rectus recession, thus rendering some patients at risk for developing anterior segment ischemia. The results of this study are encouraging; injecting the medial rectus muscle with botulinum toxin at the time of surgery can replace the need to recess the muscle and reduces the risk of anterior segment ischemia. The author hopes this study will encourage others to consider this procedure.

Keywords: Abducens, Sixth, Nerve, Palsy, Transposition, Surgery, Botulinum.

Introduction

Abducens sixth nerve palsy may be congenital or acquired. Persistent, isolated, congenital sixth nerve palsy is extremely rare, however, newborns may have a transient sixth nerve palsy that resolves spontaneously over few days to few weeks.

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A common cause of isolated acquired sixth nerve palsy in early childhood is post viral inflammatory neuropathy, which may occur in 1 to 3 weeks after a viral illness, an immunization or spontaneously without obvious cause. Improvement usually occurs within 6 to 10 weeks^[1].

After viral or idiopathic causes, the next most common causes of acquired sixth nerve palsy in children and young adults include closed head trauma and intracranial neoplasms (especially glioma and medulloblastoma). Neuroimaging is indicated for acquired sixth nerve palsy if the palsy does not improve rapidly, or if other neurological signs are present. Other causes of acquired sixth nerve palsy include Gradenigo's syndrome, meningitis, myasthenia gravis and cavernous sinus disease.

Sixth nerve palsy is typically associated with limited abduction and an esotropia that increases upon gaze to the side of the palsy. On attempted abduction, there is relative lid fissure widening as both the medial and lateral rectus muscles are relaxed on attempted abduction and the posterior orbital pressure proptosis in the eye. While in Duane syndrome type 1 there is limited abduction associated with lid narrowing and globe retraction on attempted abduction. Mild sixth nerve paresis may allow relatively good lateral rectus function and show only a trace limitation of abduction. These patients, however, will have a pattern of divergence paresis with an esotropia that is greater in the distance than at near, the divergence paresis pattern should alert the examiner to the possibility of a sixth nerve paresis^[1].

Initial therapy of traumatic or vascular sixth nerve palsy is observation for 6 months while monitoring the patient for spontaneous recovery. Spontaneous recovery of traumatic sixth nerve palsy is approximately 80% for unilateral cases and 40% for bilateral cases^[2]. A complete palsy at the initial presentation and bilateral involvement indicates a poor prognosis for recovery^[3]. During the observation period, alternate monocular occlusion or press-on prisms can be used to eliminate double vision (diplopia) if a face turn does not allow fusion.

After the 6-months observation period, lateral rectus muscle function should be evaluated, as this is critical for determining the surgical plan and often a vertical rectus muscle transposition procedure is indicated. This procedure is usually accompanied by ipsilateral medial

rectus muscle recession, which can be a high risk for anterior segment ischemia. Thus, this study was designed to eliminate the need for the medial rectus recession and the complications associated with it.

Materials and Methods

This is a prospective multicenter study in which 14 patients were included. Ages were between 6 to 76 years (mean = 42.4).

All the patients had sixth nerve palsy with causes varying between congenital sixth nerve palsy, closed head trauma and intracranial neoplasms.

The lateral rectus muscle function was assessed by saccadic velocity testing and the active forced-generation test. All patients had saccadic velocities less than 60% of normal.

Patients excluded from the study had either orbital rim fractures proven on CT scan or had saccadic velocities more than 60% normal. In total, 7 patients were excluded from the study.

All the surgeries were performed by one surgeon and were done at least 8 months after the onset of the condition.

After gaining the appropriate informed consents from the patients themselves or the parents of children, all patients underwent full tendon transposition of the superior and inferior rectus muscles to the lateral rectus muscle insertion. The surgeries were performed through two fornix approach incisions located in-between the superior and lateral recti and the inferior and lateral recti, respectively. Muscles were symmetrically transposed and sutured to the sclera using 6-0 Vicryl sutures. 8-0 Vicryl sutures were used for suturing the conjunctival incisions.

Then, the ipsilateral medial rectus muscle was injected with 7.5 units of botulinum toxin through a tranconjunctival approach.

Results

Follow-up of the patients post-operatively ranged between 6 and 18 months, which allows for enough time for the effect of botulinum toxin to disappear.

All patients had exotropia in the immediate post-operative period.

Two patients had a significant esotropia (more than 8 prism diopters) that appeared between 6 to 9 months post operatively and were advised to have a recession surgery for the ipsilateral medial rectus muscle.

Table 1 shows the details of each patient with every clinic follow-up.

Table 1. Patient details with every clinic follow-up.

Patients	Age	Pre-op Deviation	Post-op (Months)			
			3	6	12	18
1	6	40	XT = 25	XT = 10	-	-
2	10	45	XT = 20	XT = 5	ET = 5	-
3	12	35	XT = 10	Ortho	Ortho	ET = 4
4	40	30	XT = 15	Ortho	-	-
5	40	40	XT = 25	XT = 12	XT = 4	ET = 4
6	44	45	Ortho	ET = 10	ET = 20	ET = 20
7	45	65	Ortho	ET = 4	ET = 12	ET = 25
8	50	35	XT = 20	XT = 10	Ortho	ET = 5
9	51	40	XT = 25	XT = 10	ET = 5	ET = 5
10	52	30	XT = 10	Ortho	-	-
11	57	25	XT = 15	Ortho	-	-
12	58	35	XT = 15	Ortho	Ortho	-
13	62	40	XT = 20	XT = 5	Ortho	ET = 6
14	67	45	XT = 25	XT = 12	-	-

Discussion

Botulinum toxin has been previously used alone to treat sixth nerve palsy.

It was advocated to be given into the ipsilateral medial rectus muscle in order to prevent secondary contracture of the muscle and increase the chances for recovery^[4,5].

Botulinum toxin paralyzes the muscle for 3 to 6 months, thus preventing contracture. The hope is to prevent secondary contracture of the medial rectus muscle that will increase the chances of recovery without strabismus surgery. Hence, the use of botulinum toxin remains controversial^[2,6].

Holmes *et al.*, in a prospective multicenter study of acute traumatic sixth nerve palsy or paresis, reported that patients treated either with botulinum toxin or conservatively had similarly high rates of recovery^[2].

It should be noted that after a botulinum toxin injection into the medial rectus muscle for a complete sixth nerve palsy (without transposition surgery), both the medial and lateral rectus muscles will be paralyzed, resulting in essentially no horizontal movement of the paretic eye. Therefore, the patient should be warned that the paretic eye may have decreased movement after the injection. In addition, the surgeon should be aware that the effects of botulinum toxin can last more than 6 months, and surgery should be delayed until the botulinum toxin has dissipated^[7].

Transposition procedures are one of the best options for treating abducens nerve palsy. It acts by moving innervated vertical rectus muscles to the lateral rectus insertion as to provide lateral force. Patients with significant residual paresis almost always require an ipsilateral medial rectus recession to reduce adduction forces.

The vertical muscles provide substantial circulation to the anterior segment. Older adult patients, especially those with arteriosclerotic disease or hyperviscosity syndrome (HVS), are at risk for developing anterior segment ischemia after vertical recti transposition and medial rectus muscle recession.

Therefore, not operating on the medial rectus and replacing that with botulinum toxin injection reduces the risk of anterior segment ischemia.

This method also reduces the risk of over correction, which may need a further corrective procedure in the future.

The results obtained in this study are encouraging as to continue with the procedure. It controls the esotropia resulting from the sixth nerve palsy and significantly improves the abduction capability of the affected eye.

As this condition is relatively uncommon, the author hopes these results encourage other strabismologists to consider this procedure and recommends a bigger multicenter study to be carried out with longer follow-up times.

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علاج شلل العصب السادس بواسطة تغيير موضع عضلات العين العمودية ووضعها بجوار العضلة الأفقية الخارجية مع حقن العضلة الأفقية الداخلية بالبوتكس

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المستخلص. علاج شلل العصب السادس بواسطة تغيير موضع عضلات العين العمودية ووضعها بجوار العضلة الخارجية مع حقن العضلة الداخلية بالبوتكس. هدف هذا البحث هو البحث عن بديل لإجراء عملية لعضلة العين الداخلية مع العضلات العمودية، لتجنب المريض خطورة إصابة مقدمة العين بنقص في دورتها الدموية، حيث أن مقدمة العين تعتمد على الأوردة القادمة من العضلات للترود بالأكسجين. تمت دراسة ١٤ مريضاً، وإجراء عمليات جراحية لهم بهذه الطريقة، وتمت متابعتهم لمدة تتراوح بين ٦-١٨ شهراً. اثنان فقط من المرضى احتاجوا إلى عملية جراحية إضافية لعضلة العين الداخلية.